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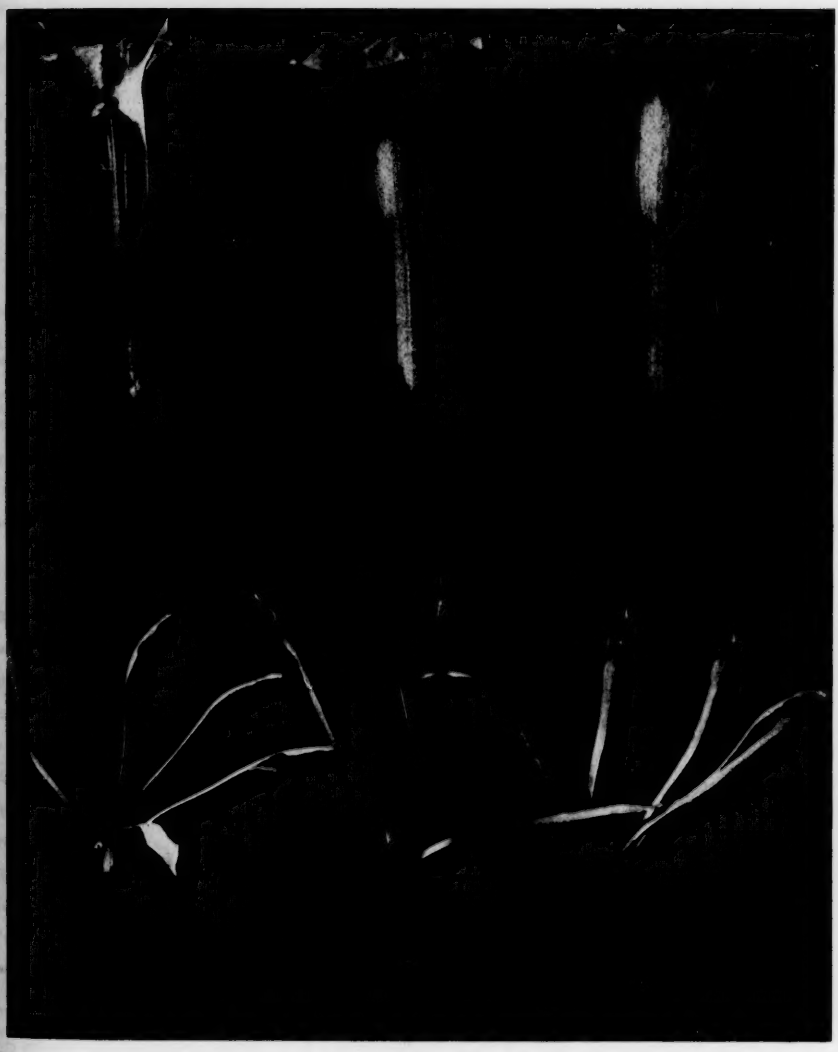

SPLIT FLOWER TOBACCO

(SEE PAGE 171)

Telomeres of Drosophila — Dwarf and Hairless Cattle

VOLUME 41 JULY, 1950 NUMBER 7

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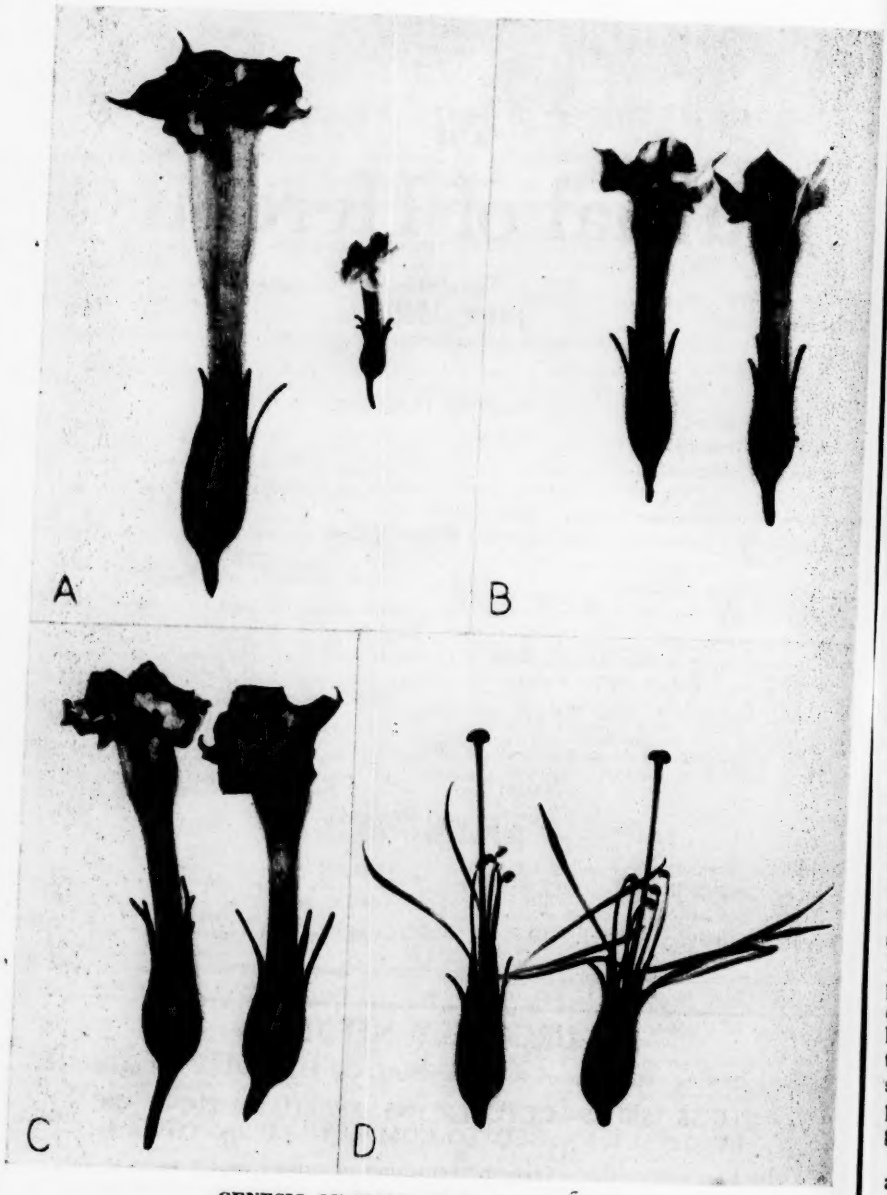


GENESIS OF MALE STERILE TOBACCO

Figure 1

In a cross between two species of tobacco, *Nicotiana tabacum* × *N. debneyi*, shown at *A*, the plants grew for a time, but only one plant in a thousand survived to maturity. The F_2 plants proved to be slightly self-fertile and were tetraploid, with a complete set of both *tabacum* and *debneyi* chromosomes (*B*). When these tetraploids were backcrossed to *N. tabacum*, the blossoms most often produced were like those shown at *C*, with some split flowers (*D*). Most of the plants with normal corollas were male sterile, as were all of the split blossoms. After two more generations of backcrossing to *N. tabacum*, most of the plants produced flowers like those shown in *D*. These male sterile types may be of value in producing hybrid seed, or to eliminate the need of topping tobacco plants in the field.

MALE STERILE TOBACCO

E. E. CLAYTON

U. S. Department of Agriculture, Beltsville, Md.

THE discovery of male sterile individuals in a normally fertile population has been reported many times. Some outstanding cases are Rhoades[3] with corn, Jones and Clark[1] with onion, and Owen[2] with sugar beet. In each case the male sterility was attributed to a special type of cytoplasm and one or more genetic factors. In no instance, however, was there any indication as to the possible source of either cytoplasmic or genetic factors. The present report traces the complete evolution of a male sterile condition in tobacco (*Nicotiana tabacum* L.)

The work began with an interspecific cross—*Nicotiana debneyi* Domin. × *N. tabacum*. Seed was readily obtained and it germinated freely. Most of the seedlings grew a few weeks and then died. About 1 in 1000 survived and produced vigorous plants. These blossomed freely and with self pollination produced a few seed. An F_2 population of some 60 plants was grown. All F_2 plants were very similar in general appearance and they were freely self fertile. These plants were proved to be tetraploids with a full set of *debneyi* and *tabacum* chromosomes (n=48). Pollen smear* examinations showed no pairing between any of the *tabacum* and *debneyi* chromosomes. These tetraploid lines were grown and observed through three generations and they remained stable. They served as the starting point for the following backcross program.

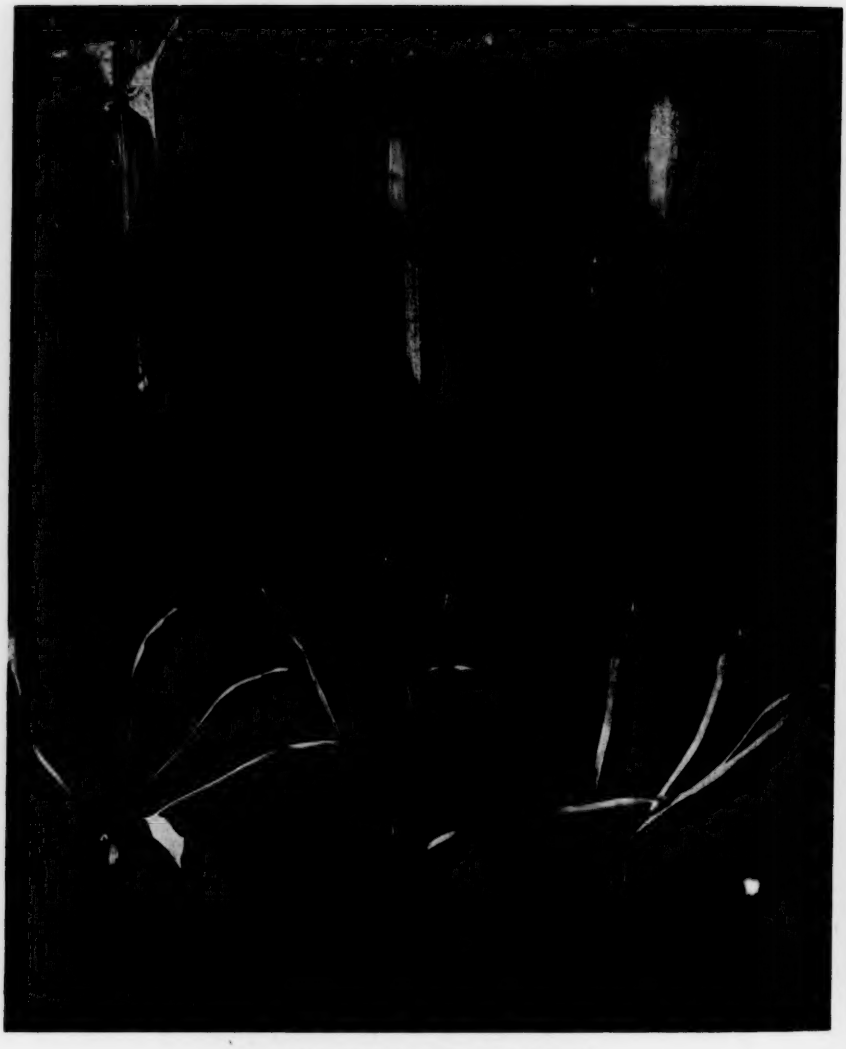
BC^1 was produced using the tetraploid as the female parent [(*debneyi* × *tabacum*) × *tabacum*]. Thee plants greatly resembled the *tabacum* parent. The anticipated chromosome situation for BC^1 was 24_{II} *tabacum* and 24_I *debneyi* chromosomes. Pollen smears indicated approximately this situation with some plants having the same 24 pairs and as few as 20 haploid chromosomes. About ten percent of BC^1 plants were completely sterile, 80 percent were self sterile, but set seed freely with *tabacum* pollen, and 10 percent were both self and cross fertile. A number of the sterile plants showed a peculiar blossom abnormality that will be referred to as split blossom (Figure 2). The normal *tabacum* corolla was divided to form five narrow petals. There was much irregularity as to the degree of splitting. However, all plants showing this character formed no viable pollen so far as could be determined.

BC^2 was produced again using *tabacum* as the pollen parent. The plants grew vigorously. The theoretical chromosome situation was 24_{II} *tabacum* and 12_I *debneyi* chromosomes. Actual counts showed 33 to 38 chromosomes at MI, with mostly the expected 36. These counts were obtained from the very few plants that produced viable pollen. Out of a group of 155 plants grown to maturity, 90 showed the split blossom character in part or all of the flowers, and all of these plants were completely male sterile. The remaining 65 had normal appearing blossoms, but all but a few of the group were also male sterile.

BC^3 [(D × T × T × T) × T] was produced by again using *tabacum* as the pollen parent. Some 8729 plants of BC^3 were grown to maturity. All but three showed the split blossom character, and all, including the three with normal appearing flowers, were completely male sterile.

In summary then the *debneyi* was the female and *tabacum* the male in the original cross. Blossoms were normal in appearance, but fertility was low. The F_2 plants were tetraploid, had normal blossoms, and were fertile (Figure 1*B*). BC^1, with *tabacum* con-

*The writer is greatly obligated to Dr. H. H. Smith, formerly with the U.S.D.A., for making the examinations through BC^2.



DETAILS OF FLOWER STRUCTURE

Figure 2

The male sterile "split flowers" obtained in backcrosses of the *tabacum* × *debneyi* hybrid are entirely devoid of anthers. The stigmas are normal, and seed is produced when *tabacum* or *debneyi* pollen is used.

tinuing as the male, showed a mixture of sterility and fertility, but with 90 percent of the plants male sterile (Figure 1*C*) and a few having the split blossom character. BC^2 was about 97 percent male sterile and over half the plants were split blossom. BC^3 was 100 percent male sterile and almost 100 percent split blossom (Figure 1*D*). Beginning with BC^4 and continuing without further change through BC^{10} all plants were male sterile and split blossom.

Thus the male sterile condition appeared with the first reduction in the number of *debneyi* chromosomes, — at BC^1. It was complete at BC^3 at which time the count of *debneyi* chromosomes had been reduced to approximately six. This material is now in the BC^{10} generation and all *debneyi* chromosomes have long been completely eliminated. The three varietal lines established are identical in appearance to the pollen parent variety except that they are male sterile and split blossom. The appearance of these lines has remained unchanged for a number of generations.

Returning now to the BC^2 stage of this work, it was at this time that the importance but not the cause of the male sterility problem was recognized. It was reasoned that this might be associated with the loss of *debneyi* chromosomes. Also, since *tabacum* had been the male parent throughout, it might be that the *debneyi* cytoplasm transmitted by the female gamete at each step was a factor. The validity of both hypotheses was tested as follows:

First, male sterile BC^3 plants were fertilized with *debneyi* pollen. The progeny showed greatly increased fertility. A number were freely self fertile. This was in contrast to the BC^3 population resulting from *tabacum* pollen, which was completely male sterile. Thus, increasing the number of *debneyi* chromosomes restored fertility and hence reversed the trend toward male sterility.

Second, BC^2 plants, that were not completely male sterile, were used to produce a reciprocal $B'C'^2$, with tabacum serving as the female parent. This $B'C'^2$ population had no split blossom male sterile plants. Examinations at MI showed 24_{II} *tabacum* and 4 to 11 *debneyi* chromosomes. Some of these plants were later backcrossed again to *tabacum* and, regardless of which direction this cross was made, no split blossom, male sterile plants have been found in the progeny. Thus back-crossing with *tabacum* could be continued without male sterility if the *tabacum* was used as the female at BC^2, or presumably any time before male sterility became complete.

An entirely separate series of male sterile tobacco types has been produced by crossing *N. megalosiphon* Heurck & Muell.-Arg. with *N. tabacum;* the *tabacum* serving as the male parent in the original cross and in subsequent backcrosses. The F_1 plants were completely male sterile, but set a few seed with *tabacum* pollen. BC^1 showed some plants with the normal tubular corolla and others with the split blossom. In BC^2 all plants showed the split blossom character with one exception. This plant had the normal tubular corolla, but the stamens were completely aborted. The progeny from this plant in BC^3 showed the same blossom type, and since then seven additional backcrosses have brought no change. The same three male sterile varieties —flue-cured, burley, and broadleaf have been established, giving an apopetalous (split corolla) series derived from the *debneyi* cross and a sympetalous (tubular corolla) series derived from the *megalosiphon*. The male sterile types and the normal, fertile pollen parent varieties are identical in appearance.

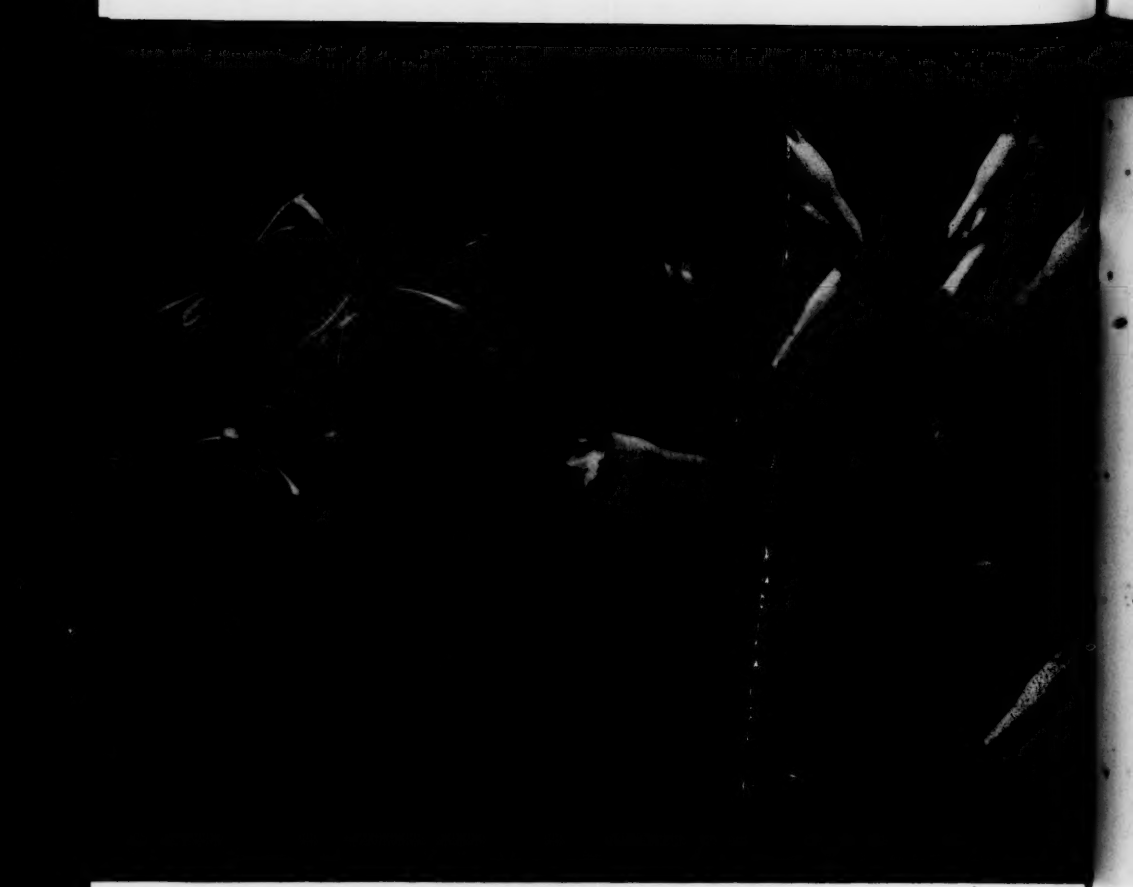
In 1948 and 1949 field plantings were made of the six male sterile lines representing separate varieties. Height and weight measurements were made and the 1949 data are given in Table I.

The data on weight and height as well as other observations and measurements indicated that the male sterile condition was not associated with any measurable reduction in plant growth.

TABLE I.—Comparative growth of male sterile and normal fertile tobacco—1949

Line	Plant Weight				Plant Height			
	Plot 1 oz.‡	2 oz.	3 oz.	Mean oz.	Plot 1 in.	2 in.	3 in.	Mean in.
Pa. Broadleaf	60.3	51.0	62.3	57.8	48.5	51.6	53.5	51.2
Male sterile (SB)* Brdlf.	61.7	60.7	82.7	68.3	49.6	49.3	57.7	52.2
" " (M)† "	51.7	81.0	82.3	71.6	52.6	58.5	56.7	55.9
402 Flue-cured	75.7	73.7	76.6	75.3	64.5	68.5	69.0	67.3
Male sterile (SB) Flue-cured	72.3	66.0	88.7	75.6	63.5	63.0	71.8	66.1
" " (M) " "	71.3	73.7	74.3	73.1	65.6	66.6	68.0	66.7
Ky. 16 Burley	60.3	90.7	87.7	79.5	58.6	63.1	63.8	61.8
Male sterile (SB) Burley	77.7	93.0	83.0	84.5	58.5	66.1	66.1	63.5
" " (M) "	66.0	71.7	74.6	70.7	56.1	70.6	64.5	63.7

* The apopetalous series from the *debneyi* cross.
† The sympetalous series from the *megalosiphon* cross.
‡ Averages from three plant plots.



SPLIT AND NORMAL FLOWER HEADS

Figure 3

The split blossom deviation is only produced in hybrids in which the *tabacum* parent used in making the backcross is the male. In reciprocal crosses in the early generations, when hybrid plants are used as the male parent, male sterility does not occur.

It was also of interest to determine how seed production by male sterile and normal fertile blossoms compared. The test was conducted in the greenhouse and all blossoms were hand pollinated (see Table II).

It is evident that the ability of the male sterile plants to produce seed, when fertile pollen was applied, had in no way been reduced.

Discussion

This study has shown that when *N. debneyi* was crossed with *N. tabacum*, and the latter species was continued as the pollen parent in successive backcrosses, male sterility was the result. The development of this sterility was gradual. Some sterility was apparent at BC^1 and all progeny were completely male sterile at BC^3. A complement of 24_{II} *tabacum* and 4 to 7_I *debneyi* chromosomes was found at this stage. To eliminate male sterility

TABLE II.—Seed production by male sterile and normally fertile plants

Line	Pod	— Seeds per pod — 1 no.	2 no.	3 no.	Mean no.	Wt. of 1000 seed gm.
Pa. Broadleaf		2295	2793	1886	2325	0.08
Male sterile (SB) Brdlf.		2938	2220	2511	2556	0.055
Male sterile (M) Brdlf.		1241	2607	2223	2027	0.07
402 Flue-cured		1676	1506	1800	1667	0.08
Male sterile (SB) Flue-cured		1860	3024	1657	2180	0.07
Male sterile (M) Flue-cured		1426	2687	2994	2369	0.08
Kv. 16 Burley		2100	1766	2190	2019	0.06
Male sterile (SB) Burley		2054	2566	1822	2147	0.05
Male sterile (M) Burley		2319	2671	3253	2748	0.06

it was merely necessary to use *tabacum* as the female not later than BC^2. It would appear thus that when the *debneyi* was the female in the original cross, and the hybrid plants were used as the female in subsequent crosses, then the *debneyi* cytoplasm was continued through the female gamete. As *tabacum* chromosomes were substituted for *debneyi*, male sterility increased and became complete after about seven-eighths of the *debneyi* chromosomes were eliminated. There was no indication that this sterility was associated with any specific genetic factor. Rather it appeared that the sterility was an indicator of incompatability between *debneyi* cytoplasm and *tabacum* chromosomes. Since the male gamete is supposed to contribute some cytoplasm, it might be supposed that by successive backcrosses repeated increments of *tabacum* cytoplasm would be added and hence ultimately normal fertility would be reestablished. However, the material has now been carried through BC^{10} without any change and presumably still has *debneyi* cytoplasm, plus the complete *tabacum* genome. These results were repeated with the *N. megalosiphon—N. tabacum* cross.

These findings have interest in several directions. First, in undertaking the transfer of disease resistance genes from a wild to a cultivated species it is frequently desirable to use the wild species, and later the hybrid selections, as the female, and the cultivated species as the male. With either of the crosses reported here this would result in the loss of all lines at or before BC^3 due to complete and irreversible male sterility. This danger may be removed by using the cultivated species as the female parent not later than BC^2.

Male sterile tobacco may have practical value in the production of hybrid seed. Also, it is possible that with some types of tobacco, male sterile crops might not require topping, since a primary purpose of topping is the elimination of seed production.

Summary

Nicotiana debneyi and *N. tabacum* were crossed and the progeny systematically backcrossed with *N. tabacum* serving as the male parent throughout. The process was repeated with the *N. megalosiphon—N. tabacum* cross.

Male sterility appeared in BC^1, increased in BC^2 and was complete in BC^3.

The gradual development suggested progressive incompatibility between cytoplasmic and nuclear constituents.

At BC^3, the level at which male sterility became complete, the average chromosome situation was *N. tabacum*—24_{II}, *N. debneyi* 4_I to 7_I.

Male sterility remained complete through BC^{10}.

The development of male sterility was prevented by using *tabacum* as the female parent at BC^2; or by using *debneyi* as the male parent at the same BC^2 level. The former procedure combined *tabacum* cytoplasm with *tabacum* chromosomes and the latter combined *debneyi* chromosomes with *debneyi* cytoplasm.

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American Publisher for Population Genetics Book

An Introduction to Population Genetics, by Ching Chun Li (reviewed in the June, 1949 issue of the Journal of Heredity), originally published in China, may now be obtained from Oregon State College Cooperative Association, P. O. Box 491, Corvallis, Oregon.

The price is $5.00 per copy postpaid.

CLOVER-LEAF TONGUES*

DAVID D. WHITNEY

LIKE MOTHER, LIKE SON
Figure 4

SOME of the tongue-movement patterns in man are inherited and others are learned. Recently Cook[1] published a note on a double-roll pattern and Hoch[2] on a triple-roll—a clover-leaf or trefoil pattern. Hoch lists three cases of individuals with a clover-leaf pattern but none of them has relatives showing it.

As a result of a small item on tongues in a popular magazine, there has come, from various parts of the country, a score or more of letters from persons describing unusual tongue patterns in their families.

The clover-leaf pattern was described in four of these families. The first family consisted of a mother and her only child (Figure 4). In the second family the same pattern occurred in the mother and one of her two children; in the third family, in the mother and one of her two sons. In the fourth family this pattern occurred in three generations but not in a direct line. The grandfather and a sister had the pattern but another brother did not. The grandfather had three daughters, two of whom have this pattern but they have no children. The other daughter who does not have the clover-leaf tongue has a son and a daughter and the daughter has the clover-leaf tongue.

A copy of the photograph by Hoch was sent to each family. Some individuals stated that they could duplicate it, some stated that they could make better clover leaves, as shown in Figure 4, while others said that they could not fully duplicate it but were able to approach it. It is evident that there is considerable variation in one's ability to make this pattern. Some think that they have learned it (as the son in Figure 4), but none of his playmates could learn it, whereas others cannot recall the time when they could not make it.

It is suggested from the study of these four families that the clover-leaf pattern could be a dominant trait with reduced penetrance.

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*Studies from the Department of Zoology, University of Nebraska, No. 246, with aid of grant from the University of Nebraska Research Council.

DWARFISM

An Hereditary Defect in Beef Cattle*

LESLIE E. JOHNSON, G. S. HARSHFIELD AND WILLIAM MCCONE†

South Dakota Agricultural Experiment Station

THE occurrence of dwarf calves has been quite frequent in many Hereford herds during recent years. These calves usually are rather thick and blocky at birth and many of them appear to possess the type and conformation indicative of developing into highly-prized show animals. However, all of these individuals fail to develop normally and soon show definite signs of dwarfism. Many of them die before reaching one year of age.

Breeders have attributed the defect to both nutrition and heredity. The number of calves occurring per herd has been relatively small, and few breeders discriminate against the sires and dams that produce such calves.

Description

This defect can best be described as "dwarfism." In general it appears to be a normal or physiological dwarfism in which the body dimensions are about in the same proportion as those of normal cattle with the exception that the dwarfs are slightly wider and blockier. The dwarfism is present at birth, but differences between dwarf calves and normal calves become more noticeable with age. It can be comprehended best from the pictures shown in Figures 5 and 6.

The defective calves are rather easily identified at birth by breeders who have had experience with the defect. First-calf heifers appear to have more difficulty in giving birth to the defective young than normal young, because of the width of body. Some calves are born dead due to calving difficulty. Slighty bulging foreheads are common, but this alteration is not extreme and may not be present.

At three to four months of age the calves show marked dwarfism and some begin to have extended paunches. None of the defective calves lived more than two years in the herd studied. (Two calves were killed for post mortem examinations.) Often the calves die before reaching one year of age. One animal lived to be approximately two years of age. This was a female and she came in "heat" before her death. She died of bloat while grazing on brome-alfalfa pasture.

The greatest number of the post mortem examinations of dwarf calves was made within a few days after birth. Two calves were autopsied at approximately one and one-half to two years of age, respectively.

Grossly, there was little to be noted in the younger calves on autopsy. One calf examined ten days after birth had never stood. The flexor tendons of the legs were contracted. On removal of the brain, the lateral ventricles contained more than the normal amount of fluid.

The two older calves had both exhibited chronic "bloat" for several months prior to the autopsies. One of these eventually died of bloat and the other was killed by bleeding. Although the rumen was overly distended with gas, no other significant gross lesions were noted.

Histological sections were prepared of tissues from several of the dwarf calves. Particular attention was given to endocrine organs—the pituitary, adrenals, and thyroid. Nothing abnormal could be noted in these organs. The thyroids were in an active state, with follicular cells of cuboidal shape. Colloid was of normal appearance.

The genital tract of a male dwarf autopsied at about two years of age was examined histologically. The tubules of the testicle showed very little spermatogenesis, but spermatozoa were present in the sections of the ampulla of the ductus deferens.

The literature enumerates numerous examples of hereditary defects in cattle. Summaries of lethal and semi-lethal characters are given by many [1,3,2,7]. More recently additional defects have been found and recorded. Various types of hereditary dwarfing are in-

*Approved for publication by the Director of the South Dakota Agricultural Experiment Station as Journal Series No. 233.

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DEVELOPMENT OF DWARFS
Figure 5

A—Dwarf calf, one day old. The blocky, compact type is evident at this age. *B*—Near calf is a dwarf 39 days old. The other calf is normal and is 44 days old. *C*—Dwarf heifer 14 months of age. This calf died of bloat about two months after this picture was taken. She came in heat once. *D*—Male dwarf calf six months of age. The calf shows the compact, blocky body of the dwarfs and the typical bloated condition. The recurrence of these semi-lethal dwarf Herefords is widespread and frequent enough to constitute an economic problem of growing importance.

cluded in the lists. Achondroplasia 1, 2, and 3 are among those listed. Short spine also produces a type of dwarfism. However, all of these dwarfism defects are extremely severe in the homozygous recessive, and the animals either die before birth or shortly thereafter. Mead *et al*[5] have reported a case of proportionate dwarfism in Jersey cows. Lush[4] has reported a case of "Duck-legged" cattle in Texas. These two defects appear to be conditioned by a single, autosomal, recessive gene. None of the dwarfism defects reported appears to resemble the one reported in this paper.

Occurrence

The data used in this study were all taken from a single herd of purebred Hereford cattle. Complete calving records were available for the investigation. Data from other herds were not used because the records were either incomplete or the breeders did not wish to risk publication of the analysis.

The defect first occurred in the herd studied when an attempt was made to linebreed to an exceptionally good-producing sire called RM in this report. Further attempts to concentrate the linebreeding resulted in more defective calves. This was true at two different locations. In this herd, nutrition could hardly have been a contributing factor as the defect had never been noted at either location before and it did not occur in offspring that were not related to RM.

NORMAL DAM AND DWARF CALF

Figure 6

The calf is nine months old and showing a bloated condition. The dwarf calves rarely survive to maturity. There is some indication that heterozygotes carrying the dwarf gene are intermediate in height, but the deviation is not great enough to make identification of heterozygotes possible.

Mode of Inheritance

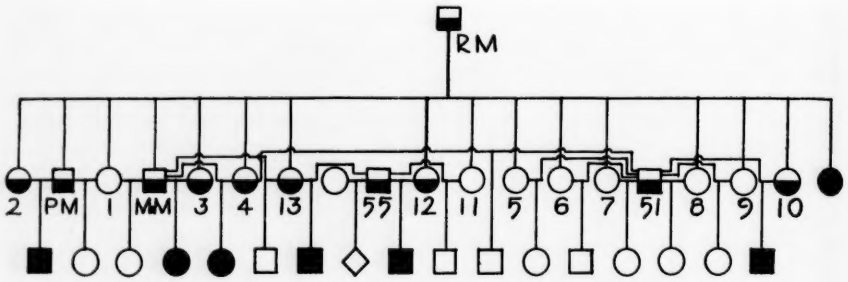
At the time this study was made, RM had produced 80 calves, 42 males and 38 females. Only one calf showed "dwarfism." This calf was a female out of a dam, RD, that had recently been added to the herd and had no direct relationship to other females in the herd. The closest common ancestor of the parents of the defective calf, RM and RD, was Prince Domino. Both parents traced to Prince Domino two or more times. Each line of descent was from four to five generations removed from Prince Domino.

Two sons of RM were used in the herd at an early date in an attempt to intensify some of the good characters of this sire. Also two other linebred Prince Domino bulls were used. All of these bulls produced some dwarf calves when mated to daughters of RM, but when mated to other unrelated females in the herd they sired only normal calves. Figure 7 shows the pedigree chart of the herd. Table I gives the individual inbreeding of the animals that produced dwarf calves and their relationship to Prince Domino.

The frequent occurrence of the defect during the period when related matings were made, its absence in unrelated matings, and its occurrence in the related mating at two locations appear to establish the fact that the defect is inherited. It also appears certain that the sire RM brought the defect into the herd except for the one cow. This cow, RD, had been recently purchased and produced three calves while in the herd. These were all sired by RM. Two of the three calves were normal. No breeding records were secured on the two normal calves. However, it was noted that the one calf was very blocky and lowset. The other was of ordinary size and conformation.

As indicated in Figure 7, both male and female dwarfs have occurred. The number of defective calves appearing in the different matings, the sexes represented, and their distribution suggest that the "dwarfism" is inherited as a simple, monofactorial, autosomal recessive. The number of animals in the different matings was too small to furnish statistically significant proof of this hypothesis.

Assuming that one-half of RM's daughters were carriers (six of the thirteen normal-appearing individuals were known carriers) and that the defect is a simple, monofactorial recessive, the mating of RM's daughters with carrier sires should produce on the average 12½ percent dwarfs. There actually occurred six dwarfs among sixteen offspring or 37½ percent dwarfs. The difference between the expected 12½ percent and the actual 37½



PROGENY OF RM DAUGHTERS MATED TO CARRIER BULLS

Figure 7

The solid symbols represent dwarfs. The other symbols all represent normal appearing animals. Symbols for the known carriers of dwarfism are half-shaded. Squares, Males. Circles, Females. Diamond, Sex unknown.

percent is somewhat large, but it is not statistically significant.

Also, it is possible that more than one-half of RM's daughters which were saved, were heterozygous. Observation has led the authors to postulate that the heterozygotes are somewhat of an intermediate in type. If this is true the selection practiced in the herd studied would have tended to eliminate the homozygous normals and to have retained the heterozygous carriers, thus producing more than 12½ percent dwarfs. In every case the cows producing dwarfs were of the intermediate, blocky, thick type. However, the old sire RM was not a particularly short, blocky bull, but he was very low set.

Discussion

A defect as serious as this "dwarfism" is certain to increase production costs in both purebred and commercial beef herds. The frequency of the gene also will increase further if the heterozygote is much preferred over the homozygote normal. If all producers used only heterozygote stock their loss would amount to one-fourth of their calf crop due to the "dwarfism." It is not at all likely, of course, that breeders could or would use only heterozygotes as breeding animals. It would seem possible, however, that three-fourths of the sires and one-third of the dams in the breed could carry the factor if breeders really tried to get an intermediate, thick type of beef animal, and if the heterozygote is an outstanding intermediate that excels those resulting from combinations of other normal genes for intermediate type. The above assumption would result in a .20 frequency of the recessive "dwarfism" gene which would produce 4 percent dwarfs on the average when the stock was being mated at random. This is probably a higher price than commercial producers will want to pay for any intermediate, thick animal. Also losses in some herds would be much higher than 4 percent as the frequency of the dwarf gene would vary from herd to herd. Purebed breeders might, of course, be able to afford to lose a rather high percentage of their calves if the carriers were able to win at many of the big shows and sales.

Regardless of the number of genes affecting the defect, it is serious enough that breeders should start culling against

TABLE I.—Inbreeding of Sires and Dams Producing Dwarfs. Also Relationship of Sires and Dams to Prince Domino

Animal	Sex	Inbreeding of Animal	Relationship to Prince Domino
RM	Male	.0891	.4123
MM	"	.0070	.2432
PM	"	.0137	.2265
51	"	.0628	.3395
55	"	.0875	.3297
BD	Female	.0049	.2091
MRD	"	.0070	.2432
PrM	"	.0318	.2394
RLA	"	.0137	.2083
RM512	"	.0070	.2432
RZ	"	.0543	.3349
RD	"	.0000	.1006

it. Certainly one should not make further outcrosses merely for the purpose of covering it up and thus spreading it further in the Hereford beef cattle population. If it is a monofactorial, autosomal recessive as indicated in the data presented, some breeders should start segregating their normal stock into lines within their herds. It would probably be too costly to sell all carriers, but records in herds possessing the defect would locate most carrier animals. For instance, if a sire produced only normal calves from ten test matings with carrier cows, it is likely that the sire is not a carrier of the "dwarfism" gene. On the average 93.5 percent of the bulls producing only normal calves in such a test would be pure for the normal genes. Not a highly accurate test could be had to select cows for a "dwarfism" free line. However, any cows that had produced a dwarf calf should be eliminated from a selected line. Breeders who establish "dwarfism" free lines will certainly have demands for their breeding animals after commercial producers have used two or three carrier bulls.

The fact that all carrier animals in this study trace to Prince Domino does not mean that all Prince Domino cattle carry the defect and that all others do not. The origin of the defective gene may have occurred considerably earlier than Prince Domino's time, or it may have occurred later in two or more separate mutations. None of our so-called lines of Hereford cattle is linebred enough to greatly increase the homozygosity of the stock. Also if the defect is inherited as a simple, monofactorial recessive, as indicated in this study, only one-half of the offspring of carrier bulls will carry the defect. Thus, many Prince Domino cattle must be entirely free of the defect unless an unusually large percentage of the heterozygotes have been saved and a large percentage of the homozygous normals culled. This appears unlikely. In attempting to purchase dwarf-free breeding stock, selection on the basis of the records of honest breeders is undoubtedly much more reliable than selection on the basis of blood lines.

Summary

An hereditary defect, "dwarfism," was observed in a herd of purebred Hereford beef cattle. The defect is evident at birth and can be classed as a latent lethal, the animals seldom reaching reproductive age. Although reliable data were available on only one herd, reports show that the defect is occurring in many purebred Hereford herds.

The frequency and distribution of the defect indicate it is inherited as a monofactorial, autosomal recessive. Observation of known carrier animals indicated that the heterozygotes, or carriers, were somewhat of the intermediate type. Intermediates in all herds however, cannot be classed as carriers as there must be other genes that produce the intermediate type beef animals.

The dwarfism reported appears to be different from other anchondroplasias reported to date. Field reports tend to indicate that the frequency of the "dwarfism" gene is increasing in the purebred Hereford population.

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TELOMERE RINGS

Figure 8

Stereoscopic photomicrographs of telomere synapsis in *D. virilis*. The ring of telomeres appears as a dark circlet at the junction of the three chromosome tips.

THE TELOMERES OF DROSOPHILA*

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THE absence of persistent simple breaks in the chromosomes of *Drosophila* following irradiation and the demonstrable existence of two or more breaks in all cases of chromosome rearrangement led Muller[8] to assume that reunion always occurred between "adhesive broken ends," rather than between free ends or by means of side attachments. Further consideration of chromosome breakage phenomena by Kossikov and Muller[7] resulted in the proposal of a rather strict bipolarity for all internal parts of the gene string and unipolarity for the free terminal genes. In the course of their investigations these authors invalidated the only case of supposed side-attachment in existence at the time (the *Pale* translocation). Following these proposals Muller[9,10,11,12] further strengthened the hypothesis for unipolar terminal genes and in the later papers employed the term "telomere" as the specific designation for such bodies. The present paper demonstrates the existence of telomeres as cytological realities in *Drosophila*.

The synapsis of the distal ends of chromosomes has been noted cytologically and described by a number of investigators since the development of salivary gland technique. Bauer[1] described the terminal synapsis as being due to some type of mutual attraction of non-specific nature between terminal chromomeres. A different view of the nature of this synapsis was presented by Prokofyeva[14], who described the presence of "inert" material in the terminal parts of the chromosome; the material was considered to contain homologous genes whose mutual attractions were the basis for the observed synapsis and for the attraction of distal chromosome regions to the inert proximal regions of the chromosomes. The frequent insertions of the ends of *X*-chromosome fragments into the chromocenter was the basis for this assumption.

Kodani[6] described the appearance of large terminal bulbs in salivary gland chromosomes which had been subjected to the action of sodium hydroxide and other chemicals and suggested that the bulbs conformed with the telomere hypothesis. These large structures can hardly be reconciled with the minute bodies which we have observed. Recently Hinton and Atwood[5] have reported detailed studies on the adhesions of chromosome tips in *D. melanogaster* and *D. pseudoobscura*. These investigators found the adhesions to be nonrandom as to the chromosomes involved and suggested the presence of a number of specific factors in the ends of chromosomes, "the frequency of the different associations being dependent on the number of the factors in common." In a further study Hinton[4], applying micromanipulation techniques to terminal adhesions, found considerable tenacity of adhering parts and observed the presence of non-stainable material at chromosome ends. On the basis of careful chromosome substitutions through hybridizations, Hinton concluded that the adhesive properties were localized at the chromosome tips, and that neither the telomere nor the heterochromatin hypotheses could account satisfactorily for the non-random nature of the observed adhesions.

Until the present writing there has been no satisfactory cytological proof of the existence of telomeres as the terminal organelles of chromosomes. Having experienced the rarity of cells

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in which these structures are clearly visible, we are not surprised at their omission from our own past illustrations and those of our colleagues. Over a period of years the present writers have kept records of *Drosophila* salivary gland nuclei which showed promise of revealing, upon further critical study, the presence of terminal chromomeres. From these nuclei, repesenting the species *melanogaster* and *virilis,* we have been able to find a number of cases in which such terminal bodies can be observed and, in *virilis,* photographed satisfactorily.

Methods

All preparations were made with standard acetocarmine smear technique, followed by permanent mounting in diaphane, and received no special staining or smear manipulations; thus our figures represent the appearance of average salivary chromosome preparation such as are available to any investigator. The microscopy entailed the use of ordinary optics: 90 × 1.4 N.A. apochromatic objectives, 15x compensating oculars, immersed 1.4 N.A. achromatic condensers, Köller illumination from a 6-volt coil filament bulb, and Wratten green filters 58 and 62. Upon completion of the drawings all details were carefully checked and confirmed with the use of a Bausch and Lomb tungsten arc lamp which provided critical illumination of high intensity; oblique lighting from several directions was employed in the final determination of telomere numbers and arrangement. The photographs were made with critical illumination from the tungsten arc. All of the photographs are stereoscopic and are mounted at average interpupillary distance of 6.5 cm.

We wish to point out that none of the illustrations* represents chromosomes which are ideal for band patterns in mapping, for in this study our objectve has been only to demonstrate telomeres. Attention will be directed toward map revisions at a later time.

Cytological Observations

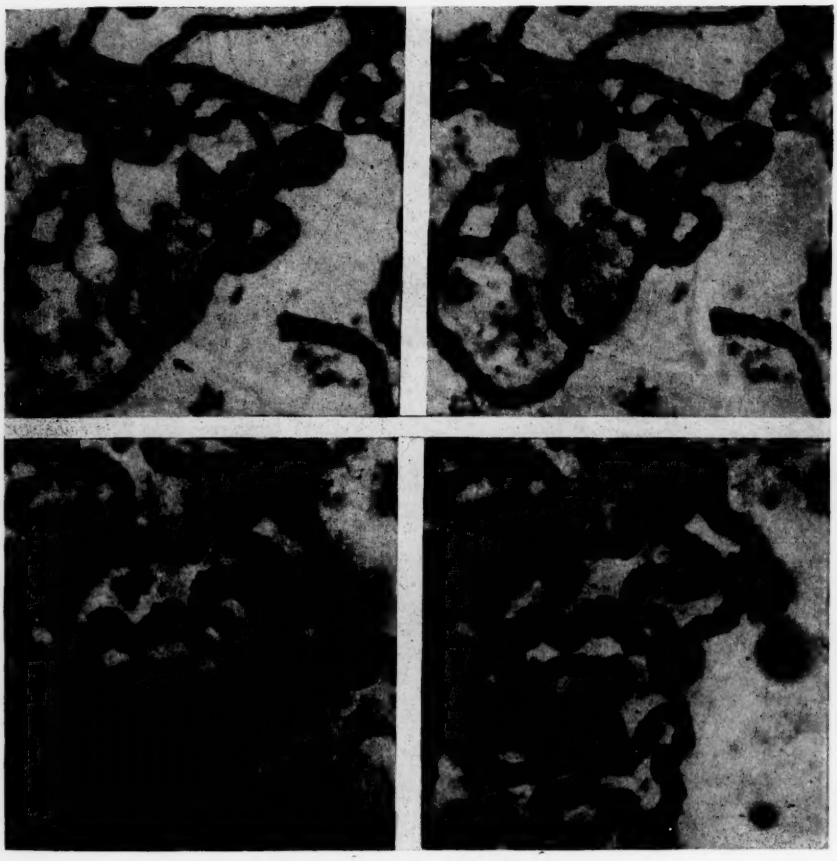
The five drawings (Figure 10) were taken from different cells which were selected for their demonstration of synapsed and of separated telomeres. Figure 10*A*, *C* and *E* show chromosomes associating in groups. The ring of chromosomes seen at the junction of the three chromosome tips (Figure 10*E*) is a portion of the telomere cluster of the cell, consisting of the telomeres of the three adhering chromosomes, illustrating the typical side-to-side attraction of synapsed telomeres and the presence of a lightly stainable border about the cluster. Telomeres are seen at comparable positions on the remaining drawings. It was not possible to determine whether the cluster found on the *X* chromosome of the Figure 10*A* and *C* represent only those of the two chromosomes illustrated in each case or whether they also include those of other chromosomes. Either condition could be possible, for observations on *D. virilis* and *D. melanogaster* indicate a tendency for the telomeres of all chromosomes to unite in a ring. This ring may be disrupted in smearing the gland or may remain intact with one or more of the chromosomes torn away.

The difficulty in finding the telomeres of a chromosome in a smear is well demonstrated by chromosome 5 of Figure 10*A*. Here the pressure exerted in smearing has pulled the chromosome from its telomeres, leaving those bodies adhering to the *X* chromosome and causing the end of chromosome 5 to spread out. A few strands connecting the chromosome with its telomeres gives the impression of a non-staining material holding the ends of the chromosomes together, as described by Hinton[4]. Two of the chromomeres of the first subterminal band of chromosome 5 have been pulled out of position and are seen lying midway along the stretched portion.

Figure 10*B* illustrates the free unsynapsed tips of two chromosomes. Numerous cells were found in which a cluster of telomeres remained on the *X* chromosome, indicating a strong tendency for the telomeres to maintain attachment with the *X*. The four spherical bodies observed on the end of chromosome 2 are interpreted as the total number of telomeres for this chromosome. Figure 10*D* shows the results of disruption of the telomere cluster. Although chromosome 2 was found at a lower position in the cell than is represented in the drawing, these three chromosome ends were probably synapsed before being torn apart in the smearing process, each carrying from the cluster a fragment which represents its own telomeres and perhaps some of those from other chromosomes. An uneven separation of telomeres would account for the presence of three in a group.

Figure 11 illustrates the telomeres of *D. melanogaster*. The number, general appearance and arrangement of the terminal chromomeres is seen here to agree with the observations made on *D. virilis*. Although

*The drawings were made with the aid of a camera lucida and represent not only our joint efforts at careful delineation but also the unbiased critical checking of Dr. Seymour Fogel, Dr. George Lefevre, Jr., Mr. D. L. Lindsley, Jr., and Mr. Jay Barton, to whom we wish to express our gratitude.



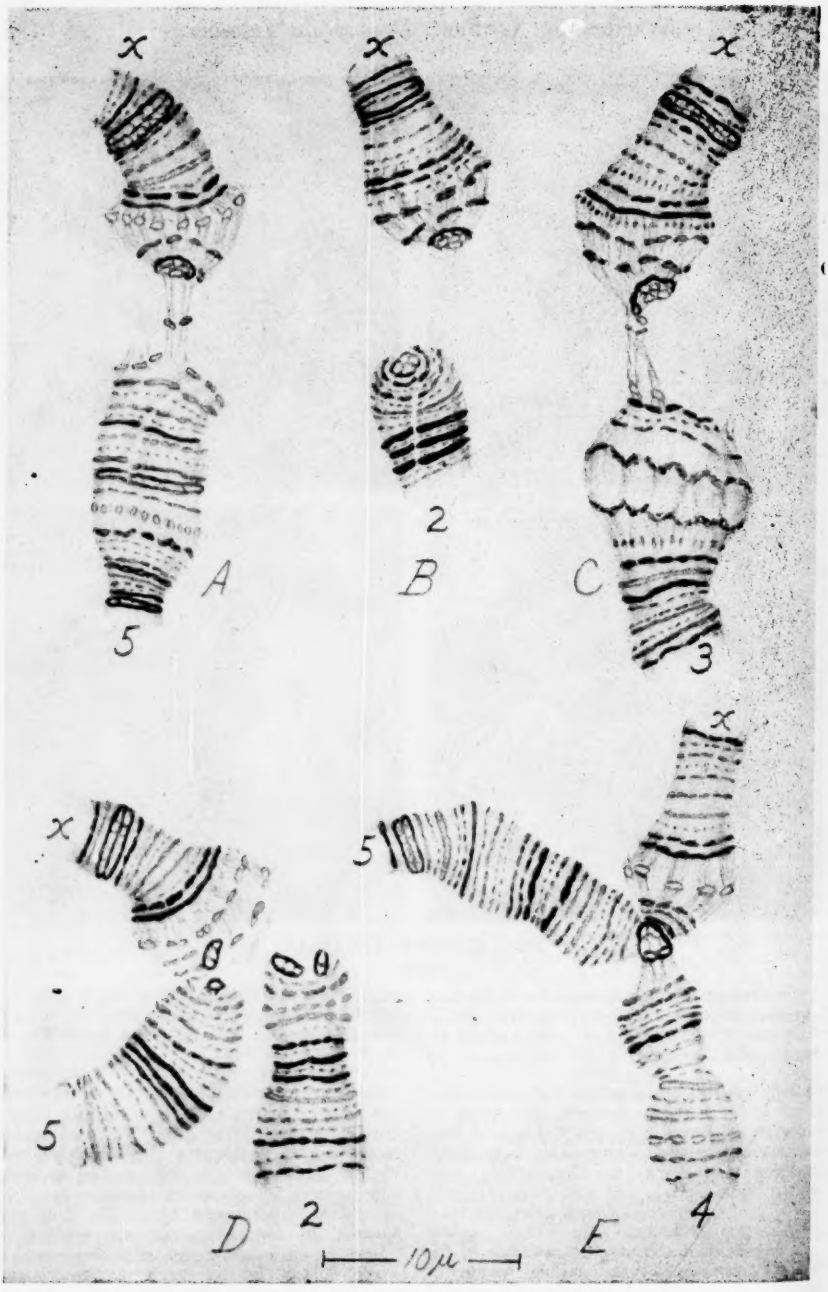
PROGRESSIVE SYNAPSIS

Figure 9

Stereoscopic photomicrographs of terminal synapsis in *D. melanogaster*. *A* shows progressive associations between chromosomes *3L*, *2R*, and *3R*, proceeding clockwise from the arrow. *B* shows chromosomes *2R*, *2L*, *3L*, and *3R* in progressive association, with the X lying free to the left of *3L*.

no cell was found in which the distal ends of all arms were in synapsis, the union of two arms as shown in Figure 11*A* was of very common occurrence. Rarer cases were found involving three arms (see Figure 9*A*), four arms (Figure 11*E*), and five arms (Figure 11*D*). In the majority of such cases the telomeres could not be resolved.

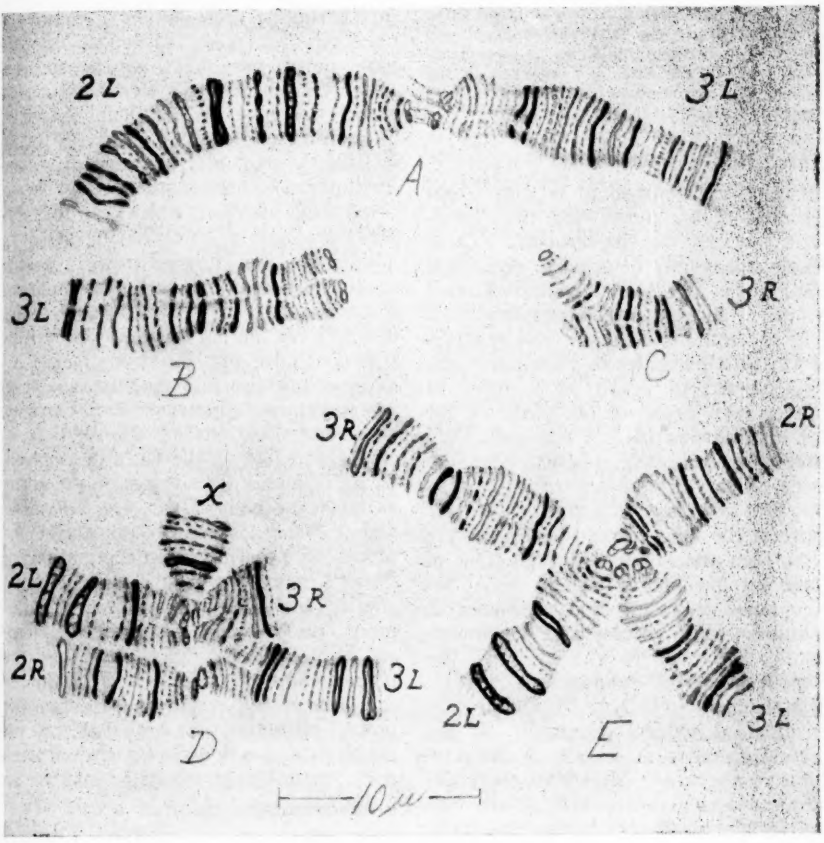
In Figure 11*A* the upper homologue of *3L* has a cluster of three visible telomeres, beyond which extend several subterminal strands of a formerly associated arm which is now torn away. The lower homologue of *3L* likewise shows subterminal strands from another arm, presumably one of the homologues of *2L*, at least one of whose telomeres has adhered to those of *3L* to make a visible cluster of three. In all instances in which more than two telomeres per homologue are visible, such strands of subterminal material can be seen as remnants of some formerly associated chromosome arm. We thus are justified in concluding that the number of telomeres on a single end of a single homologue is two in the larger salivary gland nuclei. Figure 11*B* illustrates an unsynapsed arm, *3L*, terminating in two telomeres for each homologue. Figure 11*C* was taken from the same cell and shows the unsynapsed end of *3R* with diverging tips. The two telo-



SYNAPSED AND SEPARATED TELOMERES

Figure 10

These drawings show preparations of *D. virilis*. *A*, *C* and *E* show chromosomes associated in groups. *B* shows the unsynapsed tips of two chromosomes, and *D* shows a disrupted telomere cluster. See page 185 for further details.



MELANOGASTER TELOMERES

Figure 11

These drawings show synapsis of two or more telomeres. The synapsis of two arms (*A*) was frequently observed. *B* shows an unsynapsed arm, with two telomeres for each homoloque, and *C* shows the unsynapsed arm of *3R* with diverging tips. See page 184-185 for details.

meres of the upper homologue appear as a single spherical body while those of the lower homologue, as well as several rows of subterminal bands, have been torn away in the process of smearing. Chromosomes from which subterminal bands, as well as the telomeres, have been torn away are of constant occurrence in smear preparations and increase the difficulty of finding telomeres.

The effects of telomere synapsis in *D. melanogaster* are shown in Figure 9. In the upper photograph the tips of three chromosomes can be seen at the point indicated by the arrow; *3L* lies at the bottom of the group, with *2R* and *3R* arranged in clockwise order. Each chromosome is separated terminally into its component homologues. These homologues show "progressive" synapsis of their tips, in the arrangement *3L-2R*, *2R-3R*, *3R-3L*. The lower photograph shows similar synapsis of diverging homologous tips. Proceeding clockwise from the arrow, *2R*, *2L*, *3L* and *3R* are progressively attached; the arrangement here is *2L-2R*, *2R-3L*, *3L-3R*, with *2L* and *3R* each having one homologue broken away. The missing parts may originally have been synapsed to those of the *X* which appears at the center of the photograph.

The photographs of Figure 8 present different foci and different stereoscopic views of a ring-shaped telomere cluster in *D. virilis*. Figure 10*E*, the drawing of this cell,

will serve for identification of the three chromosomes. In the middle photograph the telomeres of chromosome 4 can be seen clearly as parts of the ring, still attached to the rest of the chromosome by lightly stained stretched material.

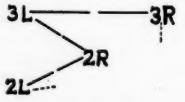
Discussion

From the figures it is clear that terminal chromomeres are real structural units of the chromosome. These bodies are readily detected in occasional ideal nuclei of *D. virilis*, the collected telomeres forming a compact ring with a lightly staining border. The synapsis of all of the telomeres in a nucleus might be expected to result in a spherical cluster such as is readily observed for the centromeres of this species[13]. We have not seen such spherical arrangement, since we have not yet attempted the study of intact nuclei. Two or more of the long chromosome arms are often seen attached to the ring. One of these chromosomes is usually the *X*, accompanied frequently by autosomes 2, 4 and 5. Chromosome 3 seldom maintains attachment to the cluster. We consider this non-random maintenance of attachment to be based upon the subterminal structural strength of the several chromosomes. The *X* chromosome, for example, shows an especially strong interstitial structure adjacent to the telomeres, always having a heavily fibrous appearance and maintaining attachment to the cluster with marked tenacity. Chromosome 3 has a puffed subterminal structure and seldom maintains attachment to the cluster. The remaining elements have intermediate strengths which we have not attempted to seriate on the basis of attachment frequencies; however, in nuclei in which the telomere cluster has been disrupted, portions of the cluster are seen attached to *X*, 2 and 5 more frequently than to 4. This view is in close agreement with that of Goldschmidt and Kodani[3], who convincingly discussed terminal adhesions as well as subterminal breakage in smearing; these authors, however, attributed adhesions to a "stickiness of the telomeres."

In the course of the present study and in the routine examination of innumerable cells in other investigations we have never seen anything to suggest the presence of inert or "heterochromatic" material at the distal ends of any chromosomes (using these terms in their cytological sense as does Prokofyeva); nor, more specifically, is there any material which can be described as similar to or having synaptic affinity for the known heterochromatin of the proximal chromosome regions. Since we have found telomeres to exist and to have mutual synaptic properties, it is concluded that insertions of the *X*-chromosome tips into the chromocenter, a very common appearance in *X*-chromosome translocations of the w^{m5} type, are explainable on the basis of synapsis of *X*-telomeres with those of the short chromosome ends of 4*L*, 4*R*, X*R*, Y*L*, and Y*R*. Since we do not feel competent to present an interpretation of Bauer's expression "non-specific, mutual affinity" as applied to terminal chromomeres, we believe that our term "synapsis of telomeres" fully describes the phenomenon, implying that all telomeres are similar and display a tendency for mutual attraction; we have not seen indications that any adjacent chromosome parts may likewise have synaptic affinity.

The stereoscopic photographs (Figure 9 *A-B*) illustrate mechanical disruption of telomere synapsis in *D. melanogaster*. In *A* the chromosome tips show the pattern

in which the lines represent diverging homologous chromosome ends. Similarly, in *B* the pattern is



with 2*L* and 3*R* each having a tip broken away. On the basis of Hinton's interpretation these configurations might be considered to indicate greater affinity between certain non-homologous ends than between homolo-

gous ends, caused by genetic factors of different parental origin. Since these cells and others illustrating progressive attachment were observed in stocks whch had long been inbred, we cannot subscribe to this explanation. Our interpretation is that the progressive terminal associations are the result of incomplete disruption of telomere synapsis, the telomeres originally having been associated closely in ring or cluster form; during the growth in diameter which characterizes salivary chromosomes the lateral expansion (reduplication) of subterminal structures separated the telomeres of homologues without disrupting relations between non-homologous chromosomes as shown in Figure 11. The remaining synapses of the telomeres on non-homologous ends are of importance in demonstrating not a repulsion of the telomeres of homologues, but the similarity of telomeres on non-homologous ends. We consider this to be strong evidence that all telomeres are fundamentally similar and probably identical.

The presence of demonstrable telomeres on the well-known distal ends of chromosomes, especially in *D. virilis*, indicates that the obscure short arms likewise terminate in telomeres. We have not been able to identify such terminal bodies in *virilis* beyond the centromere positions. Only a fragment of one of these short arms, that of chromosome 4, has been demonstrated cytologically[13].

Number of Telomeres

In all cases in which the telomeres can be distinctly seen on isolated chromosomes the number of the bodies appears to be four: one for each chromatid of the synapsed homologous chromosomes. We are therefore in agreement with Bridges[2] and Bauer[1] in their observations that salivary chromosome tips fail to show the reduplication which characterizes other portions of the chromosomes. In this characteristic of non-reduplication during salivary chromosome development the telomeres and centromeres are similar. The total telomere count in *D. melanogaster* is considered to be 32, based on the presence in each cell of four original chromatids for each pair of homologues and the failure of these chromatids to reduplicate at their ends. This number is the same for both males and females, since the *X* and *Y* chromosomes are known to have two arms. Similarly, *D. virilis* is considered to possess 48 telomeres, based on the presumption that each of the six chromosome pairs has subterminal centromere position. By way of generalization, the number of telomeres per cell in a species is twice the number of centromeres, or $2N$ where N is the total centromere number. It follows that the telomere number in cells carrying ring chromosomes, e.g. closed -*X* in *melanogaster*, is $2N$-4 for the heterozygote and $2N$-8 for the homozygote.

The reality of telomeres as the terminal unipolar bodies on chromosomes re-enforces the doubt that terminal deficiencies can be produced in *Drosophila*. Since the question has been discussed thoroughly by Muller[11,12] we shall not consider the matter at this time; we can only state that if telomeres are present in the supposed cases of terminal deficiencies, they may be demonstrable.

Summary

1. The salivary gland chromosomes of *D. melanogaster* and *D. virilis* terminate in visible specialized chromomeres, the telomeres.

2. In *D. virilis* the telomeres synapse in side-by-side manner to form a ring-shaped cluster as seen in most cells. It is presumed that the total cluster should have spherical form such as is observed for the centromeres in this species.

3. In *D. melanogaster* the telomeres can be resolved only with great difficulty and have not been observed in ring or spherical synapsis. The synapsis is usually disrupted in the preparation of smears, leaving tips, whose telomeres were formerly adjacent in a cluster, apparently adhering end to end.

4. Like the centromeres, telomeres do not reduplicate during nuclear growth, but maintain their original number of two for each end of each homologue.

5. No evidence is seen for the presence of terminal heterochromatin or inert material in the cytological sense of these terms. Attraction of the chromosome tips to the chromocentral region of the nucleus is considered to be due to the presence of telomeres on the obscure short chromosome arms.

6. There has been no indication of synaptic attraction between any terminal regions of the chromosomes other than the telomeres.

7. The subterminal structural strength of each chromosome determines the ease with which the chromosomes are separated from the cluster and from each other upon smearing.

8. Because of their marked capacity for synapsis, all telomeres of a cell are concluded to be fundamentally similar and not individually specific for the several chromosomes.

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A RECURRENCE OF THE SEMI-HAIRLESS GENE IN CATTLE*

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In the larger domestic animals, many anomalies conditioned by specific recessive genes have been discovered, particularly as a result of inbreeding. Among these, various degrees of hairlessness have been found rather frequently. Craft and Blizzard cite reports of its occurence in cattle, sheep and swine as well as in rats, mice and rabbits.

A comprehensive review of the literature indicates that in cattle there are at least five distinct recessive genes conditioning different epithelial defects. Cole[1] reported a hereditory epithelial defect in a Holstein-Friesian herd. A different congenital epithelial anomaly referred to as Epitheliogenesis Imperfecta Neomatorum Bovis was described by Hadley[3] and was discussed further by him and his co-workers in later papers. Regan, Mead and Gregory[9] reported the same condition in the University of California Jersey herd. This anomaly was also reported in a Jersey herd in Texas by Wipprecht and Horlacher[11]. The California and Texas herds were shown to be related. Mohr and Wreidt[8] in Sweden, described a different gene conditioning hairlessness in Holstein-Friesian calves (Hypotrichosis Congenta). Surrarrer[10] described another epithelial defect, apparently congenital.

Craft and Blizzard[2] studied a condition of semi-hairlessness in a grade herd of Polled Herefords in Oklahoma. It was not lethal, but the affected calves did not grow as well as those with normal hair. Hair was absent from the margin of the ears, in areas along the ventral side from the sternum to the udder, on the inside of the legs, and from areas along the side of the neck, shoulder vein, sides and thighs. The tail was almost bare and the brush hair at the tip of the tail was lacking. The bare areas of skin were pink and showed a greater tendency to wrinkle than those covered with hair. The affected skin became dry and scurfy after a few weeks. The hair appeared to shed from the follicles very early on the afflicted animals. In certain areas the hair appeared to break off relatively close to the skin. Microscopical examinations

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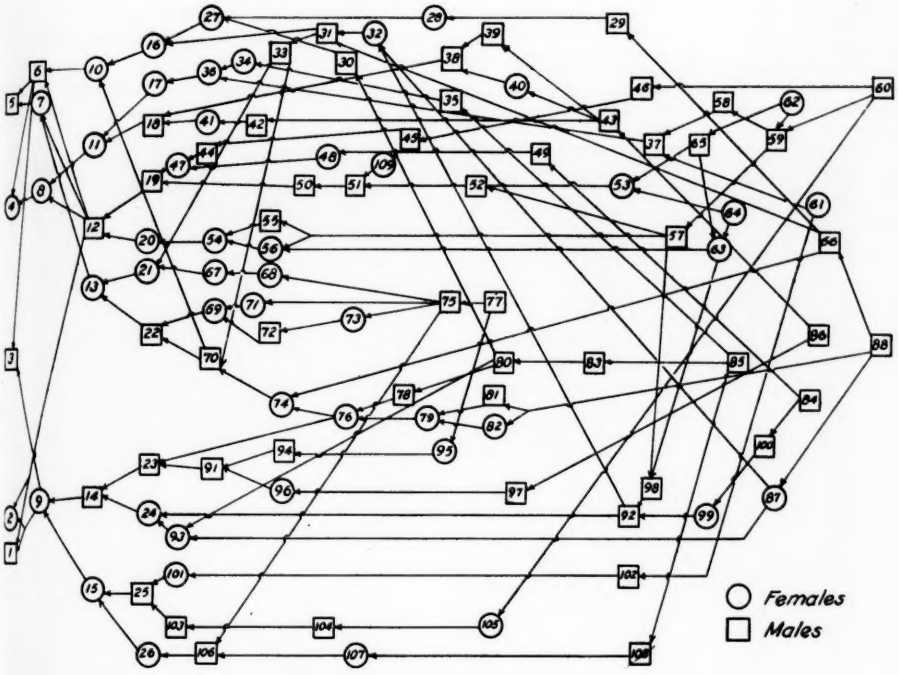

PEDIGREE OF HAIRLESS CALVES

Figure 12

Analysis of the pedigree of five hairless calves shows that they were all related, and suggests that their common ancestry traces in several lines to individuals which had already been identified as being carriers of the hairless gene.

showed that the hairs had a tendency to be somewhat flat and spiral. No constrictions or other abnormality of the hair was observed. The affected animals were wild and would not be tamed.

Analysis of observed data and results of experimental mating of stock procured from the herd, further substantiate the suggestion that the semi-hairless condition is a recessive character.

The purpose of this paper is to describe and record the appearance of a gene conditioning semi-hairlessness in a purebred herd of Polled Hereford cattle and to analyze its mode of inheritance.

Description of the Anomaly

The present case was brought to the attention of the authors by a student of the University of California whose father is a Polled Hereford breeder in California. During the period from 1943 to 1948 five abnormal calves were dropped in the herd. The first two were full sibs, a bull calf dropped in 1943 and a heifer calf dropped in 1944 (male 1 and female 2, Figure 12). The owner described the calves as having very little hair and being hard to fatten. When the bull calf was butchered, the hide was found to be exceptionally thick. There was very little internal fat. The third case, a bull calf dropped in 1947 (male 3, Figure 12), was a maternal half brother of the first two and was sired by a son of the sire of the first two. He was described as queer acting, and strayed off part of the time. He was small for his age, of undesirable conformation and appeared to have defective eyesight. The fourth calf, a heifer (female 4, Figure 12), was dropped in the spring of 1947. She did not fatten, was small for her age and had about a half coat of hair. The last calf (male 5, Figure 12), was dropped April 14, 1948. He was small with very little hair and subject to sunburn.

The last calves were both double grandsons of male 12, the sire or grandsire of the first three calves.

Unfortunately the authors have not been able to observe these animals and the descriptions are meager. The descriptions, however, seem to coincide closely with the anomaly reported by Craft and Blizzard[2]. Wildness, small size for age, breed of cattle, and the fact that the condition was not lethal are particular points of coincidence.

Analysis of Mode of Inheritance

A pedigree network giving the pertinent ancestry of all afflicted animals is shown in Figure 12. Code numbers, rather than names and registration numbers, are used.

Male 3 has two full sibs, dropped in 1945 and 1946 respectively, and male 5 has a full sister, dropped in 1947, that are normal. This evidence strongly suggests that the defect is conditioned by a recessive gene. Since the anomaly occurs in both males and females, and since phenotypically normal animals evidently are carriers, it is assumed that the caustive gene is autosomal. It also follows that all the afflicted calves must be homozygous for the gene in question and that their normal parents must be heterozygous. The heterozygosity of female 9 and male 12 is established by their homozygous offspring, male 1 and female 2. It is more reasonable to assume that heterozygous animals 6, 7 and 8 received the same gene from a common ancestor than that an identical mutation recurred three times. Male 12 and female 9 may be traced to male 60, female 62, female 64 and male 84. It appears certain that one of these common ancestors introduced the gene into the herd. Since the gene was first reported in a grade Polled Hereford herd it is probable that the animal introducing it into this pedigree is related to the animals studied by Craft and Blizzard. It would be interesting to compare the ancestry of these herds since it is possible that they are related through some common ancestor, i.e. male 60, female 62, female 64, or male 84.

Summary

A study was made of a semi-hairless anomaly which occurred in a herd of purebred Polled Hereford cattle in California. An analysis of the pedigree indicates that the anomaly is conditioned by a single autosomal recessive gene. This anomaly is probably identical with the one studied by Craft and Blizzard.

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Genetics in Japan

THE National Institute of Genetics has recently been established at Misima, Sizuoka-ken, Japan. The purpose of the institute is "to be an organ of comprehensive studies on the principle of heredity, and of providing a theoretical basis to the solution of practical problems on heredity, and also to guide and promote the genetic researches in Japan."

The field of research in the institute covers all branches of human, animal and plant genetics, either theoretical or applied.

FUZZY MICE*

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INFANT AND ADULT FUZZY MICE

Figure 13

The litter of two fuzzy and two normal mice is about one week old. The adult mouse shows the typical pelage of this type.

MANY wavy haired characters have been noted and studied in the mouse (*Mus musculus*). The amount of waving or curling of the hair varies in intensity but is consistent for each character. Further, as the animals mature and age, the waving in the coat becomes less noticeable and only the vibrissae remain conspicuously curled. This is true of rex (*Re*), caracul (*Ca*), waved-1 (*wa*-1), wellhaarigkeit (*we*) and waved-2 (*wa*-2). These characters are inherited in a regular Mendelian manner.

The present report will describe a new curly character that might be listed with the group above but differs in several ways so that it is not a mimic gene. Also the purpose of the study of this new character has been to determine if fuzzy is inherited and to ascertain the manner of inheritance and, if possible,

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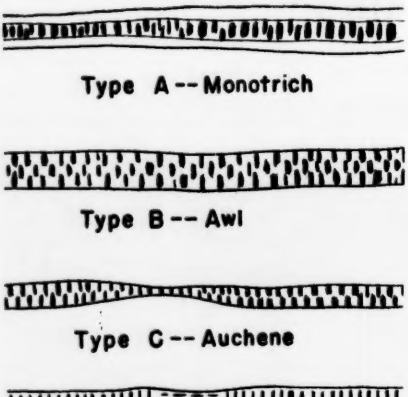


NORMAL FUR TYPES

Figure 14

The four normal hair types shown here occur in the coats of normal animals. The fuzzy mice have several other types of hair. See Figure 15.

its location on the chromosome map of the mouse.

Origin

The mutation was first discovered in 1945 in the CFW stock of Mr. F. G. Carnochan at Carworth Farms, and some of the mutant mice were sent to this laboratory for study. Studies on the action of this mutant were being made at the time of the Bar Harbor fire in 1947. The stock maintained here was destroyed. However, the gene was recovered from Dr. Clara Lynch at Rockefeller Institute, who was comparing the fuzzy character with a mutant that occurred in her mice. Since recovery of the gene, the stock has been built up and linkage tests have been made.

Breeding Data

In order to determine the mode of inheritance of fuzzy, various crosses were set up. These are summarized in Table I. Animals were classified on the basis of curly coat. Mice from six days to weaning age were classified.

Fuzzy × fuzzy produced over 100 offspring, all animals were of the fuzzy type. Fuzzy × normal produced all normal coated animals, therefore the character seemed completely recessive. Further evidence of the recessive nature of the character was shown when heterozygous fuzzy mice were mated *inter se*. Of the offspring of this cross 20.84 percent were fuzzy, a satisfactory approximation to the expected 3:1 ratio. Heterozygous fuzzy animals backcrossed to fuzzy mice gave 50 percent fuzzy, exactly the expected 1:1 ratio.

From the above data it was evident that fuzzy was a unit recessive mutation and *fz* was suggested as the symbol for the character.

Description

Fuzzy animals are easily distinguishable from normal and from other curly coated animals in the following respects: the hair is much thinner and faintly wavy, the vibrissae are not curled but seem to be very thin and faintly wavy. Animals can be classified at six days of age by the vibrissae and by

TABLE I.—Segregation of Fuzzy

Mating	Non-Fuzzy	Fuzzy	Total	% Fuzzy
fz/fz × *fz/fz*	0	102	102	100
fz/fz × *Fz/Fz*	61	0	61	0
Fz/fz × *Fz/fz*	357	94	451	20.84
Fz/fz × *fz/fz*	58	58	116	50.0

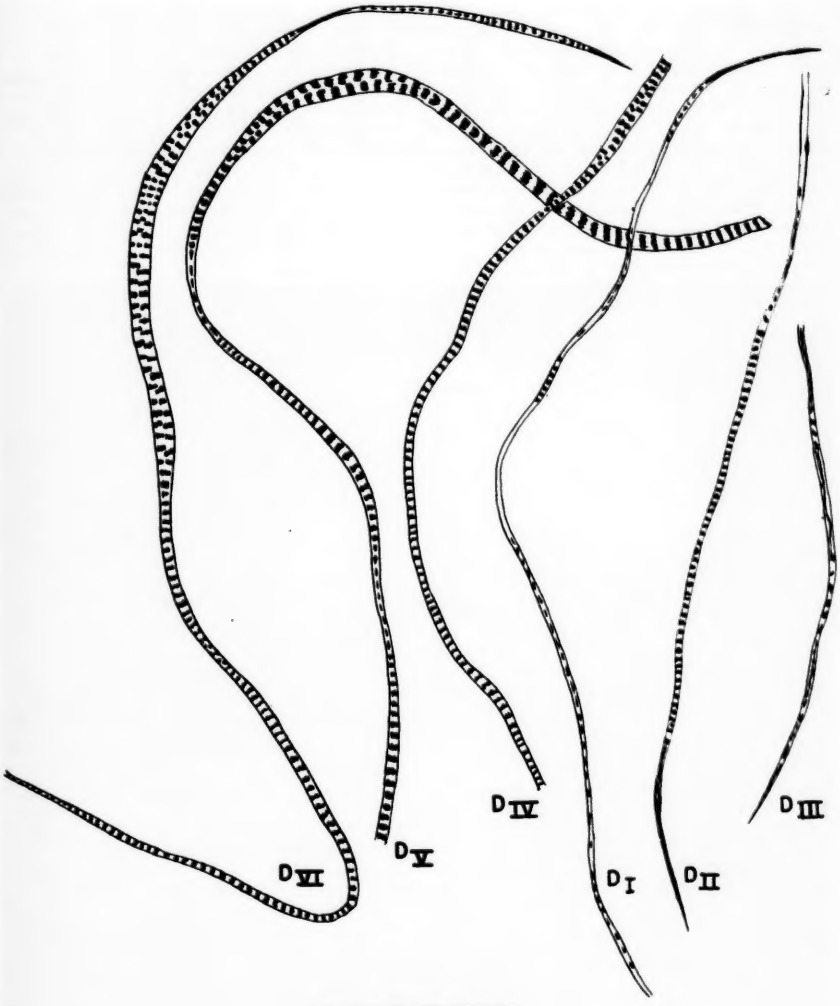
TABLE II.—Summary of results of crosses with other curly coated animals in the F_1 generation.

Mating	Fuzzy	Other Mutant	Normal	Total	% Normal
fz/fz × *Ca/Ca*	0	34	11	45	34.4
fz/fz × *wa-1/wa-1*	0	0	15	15	100.0
fz/fz × *wa-2/wa-2*	0	0	12	12	100.0
fz/fz × *Re/re*	not tested				
fz/fz × *wa/wa*	not tested				

TABLE III.—Summary of results of linkage tests

fz tested with	Linkage Group	Total No. Animals	Cross-over Percentage
**pi*	III	150	53.75
hr	III	142	60.56 ± 4.1
cc	I	65	49.23 ± 6.2
†*A*	V	92	57.46
Ca	VI	215	60.00 ± 3.3
Fu	IX	215	51.62 ± 3.3
Wv	III	215	48.83 ± 3.3
Sd		215	53.48 ± 3.3
§*f*		146	46.58 ± 4.1
sh-2	I	being tested	
wa-2	VII	being tested	
**d*	VIII	189	53.83
**b*	VIII	189	49.18
s	III	being tested	
v	X	being tested	
‡*ln*		300	41.00 ± 2.9
wa-1	XI	being tested	
§§*p*		92	41.30 ± 5.2
bt	VI	being tested	
ru	XII	being tested	
ja	XII	being tested	
m	VIII	being tested	

*data on F_2 repulsion cross ‡linkage proven
†data on F_2 coupling cross §further possible linkage



FUZZY HAIR TYPES

Figure 15

The six hair types shown here are typical of the fuzzy mouse. Type DIV to DVI are found only in standard fuzzies and heavy fuzzies.

the unevenness of the coat (Figure 13*A*). At times in the mature animals there seems to be little waving of the hair but it is much thinner than in a normal coat (Figure 13*B*).

There have been two noticeable changes in the gross appearance of the fuzzy coat from the original type or standard fuzzy. One has been an intense curling of the hairs so that it resembles persian lamb; the other combines characteristics of both the heavy coat of the persian lamb type and the kind of waviness of the standard type.

Preliminary studies* on the hair of the fuzzies indicate that they lack certain types

*Early studies of fuzzy hairs were done by Miss Claire Fulcher, a summer student at the laboratory in 1946.

of hair common to normal coats and other curly coated animals. In normal animals there are four main classes of hairs; monotrichs or Type A, awls or Type B, auchenes or Type C, zigzags or Type D and intermediates of these types, A-B, B-C, and C-D. Hairs from waved-2 animals and caracul animals were examined and all these types were present, the only difference from normal being that the hairs were waved or curled. Upon examination of hairs from fuzzy animals it was noted that other than zigzag hairs, none of the main types was present. The hairs observed seemed to be zigzag combinations, and were quite different from any described in Dry's classification of hair types.

Studies of the hairs of the three types of fuzzy have shown that each has certain types of these zigzag and zigzaglike hairs. (See Figure 15). Three types are common to all the fuzzy animals. Since they are all modifications of the zigzag or Type D hair they will be named D_I, D_{II}, D_{III}, etc. D_I hairs have numerous constrictions but at the constrictions they are lacking in septate parts. In the non-constricted portions of the hair they are septate. D_{II} hairs have no constrictions but are broader in parts than normal zigzag hairs, resembling a monotrich without the heavy cortex. The rest of the hair is like the non-constricted portion of a zigzig hair. D_{III} hairs do not really resemble any classified hair. They tend to be septate, but separation is not clear and the hair has no constrictions. The next two hair types are found only in the coats of standard fuzzies and heavy fuzzies. D_{IV} resembles the intermediate C-D type. D_V is a combination of Types B, C, and D. Part is II-septulate and it has numerous constrictions that are septate. The last type of hair found, D_{VI}, occurs only in heavy fuzzy coats. Like the intermediate A-B type it is partly III-septulate, but it is also II-septulate and septate and has several constrictions.

Although three types of fuzzy have been observed, at this time breeding tests with these types have not progressed far enough to indicate definitely whether they are alleles or the result of modifying genes.

Crosses With Other Mutants

Linkage tests were set up with many of the characters already located in linkage groups and also with various independent characters. (Table III). Wherever possible and practical, the backcross was used for classification of animals. With the curly coated characters, classification was very difficult, since it was practically impossible to decide if an animal carried both fuzzy and the other character. Therefore data with such genes were open to question and it was decided to test other characters nearest to those curly coated genes. Tests thus far have shown that fuzzy is linked with leaden (hitherto a long independent character studied by Murray), and the cross-over percent for 300 animals being 41.00 ± 2.9. Classifying data for heterozygous females separately, the cross-over percent is 49.16 ± 4.0 and with the heterozygous males the cross-over percent is 39.13 ± 4.1. If animals are classified according to the parent from which they came, the results are as follows. With *lnfz* female parent, cross-over percent is 41.48 ± 4.5 and with *lnfz* male as parent, 40.90 ± 3.7 percent. Additional linkage results will be discussed in another report.

Discussion

Mice with *fzfz* coats are easily distinguishable from normal and from other curly coated characters. Classification may be made at an early age by observing the vibrissae and the coat. There seem to be at least two types of fuzzy at the present time, the standard fuzzy and the caracul fuzzy which looks like persian lamb. While the fuzzy is quite distinct from the other curly coated characters, it is virtually impossible to classify animals that carry fuzzy and another curly coat gene. The absence of certain types of hair that are normally present in the coat of the mouse would seem to open up problems as to the significance of the lack of these hairs and the physiological basis for such a depletion.

Summary

A new curly coated character in the house mouse is described. While similar to other curly coated animals, it is easily distinguishable from them by the types of hairs present or lacking in the coat. Evidence is presented that the character is a unit recessive mutation and the suggested name for this mutation is fuzzy with the symbol *fz*.

Linkage tests with fuzzy have shown that it is linked with leaden with a cross-over percent of 41.00 ± 2.9.

The possibility of the occurrence of other alleles of fuzzy is mentioned and a description of two, caracul fuzzy and heavy fuzzy with the hair types that they carry.

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